

The information in this column is not intended as a definitive treatment strategy but as a suggested approach for clinicians treating patients with similar histories. Individual cases may vary and should be evaluated carefully before treatment is provided. The patient described in this column gave informed consent for the publication of the column.

Social anxiety disorder in schizophrenia: a neglected, yet potentially important comorbidity

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Social anxiety is highly prevalent in people with schizophrenia and can hinder functional recovery. Our patient received a diagnosis of schizophrenia at age 23. He did not respond adequately to antipsychotics during the first 2 years of illness; he experienced severe psychotic exacerbations and eventually committed a crime that was covered widely in the media. When he was 25, his psychotic symptoms responded significantly to clozapine, which he tolerated well, without any akathisia. However, he remained severely socially withdrawn. Although he first stated that the withdrawal was because people were talking about him, upon closer questioning, he revealed that he actually feared people would talk about him (rather than perceived that they did). In addition, our patient did not think that people were trying to harm him or that they had a prior knowledge of him. In fact, he feared that people would judge him and think he was a loser and that people who witnessed his strange behaviour during his psychosis would recognize him. A social anxiety disorder (SAD) was diagnosed, and a selective serotonin reuptake inhibitor (SSRI) was then introduced as a first intervention because the severity of his social anxiety symptoms (SAS) prevented him from gradual exposure. The SSRI allowed his SAS to decrease to a level that allowed gradual exposure and cognitive behavioural therapy (CBT) with a psychologist, supported by a case manager. Eight years later, our patient now works full time, lives independently and has an active social life.

A meta-analysis¹ reported a prevalence of SAD of 14.9% in people with schizophrenia, and a recent study reported a rate of 47.5% in a cohort from an early-intervention clinic.² Striking differences in rates can be observed across studies, which can be partly explained by differences in assessment methods.¹ More specifically, the 2 studies reporting the highest rates of SAD^{2,3} incorporated the probes from the Liebowitz Social Anxiety Scale (LSAS), which increases the sensitivity for detecting SAS and SAD.

Comorbid SAD is clinically important, as lower functioning and higher risk for suicide attempts have been observed in people with schizophrenia and comorbid SAD.^{3,4} Yet, SAD is generally underreported in clinical settings, probably due to difficulty in disentangling symptoms of psychosis from those of SAD. For instance, our patient's initial description could suggest ideas of reference, and his avoidance could have been interpreted as a negative symptom. However, as his social avoidance was a consequence of his social anxiety rather than lack of interest, it would be considered as a negative symptom secondary to anxiety.⁵ The validity of SAD diagnoses in people with schizophrenia is supported by several differences observed between patients with schizophrenia with and without comorbid SAD:

- distinct profiles of cognitive⁶ and social cognitive impairments;^{7,8}
- attribution biases affected specifically in patients with comorbid SAD;⁸
- higher levels of shame associated with the schizophrenia diagnosis;⁹
- lower self-esteem, and higher levels of self-blame and entrapment;¹⁰
- an insecure attachment profile and a positive history of childhood trauma;¹¹ and
- perception of lower social rank.⁶

Thus, it seems relevant to use extensive probing to identify and eventually treat SAD comorbid to schizophrenia.

Before initiating treatment for SAS, one should first determine whether the symptoms stem from psychosis or from akathisia.¹² Anxiety resulting from psychotic symptoms would require optimizing the antipsychotic treatment, whereas anxiety judged to be a behavioural manifestation of akathisia¹³ would require lowering antipsychotic dosage, switching to an agent less likely to cause this adverse effect or using add-on medication to address akathisia.

If the SAS are deemed to result from an SAD, we are aware of only 2 unblinded randomized controlled trials that specifically targeted SAD^{14,15} using CBT, suggesting that such approaches could reduce SAS in patients with schizophrenia and comorbid SAD. As for pharmacological treatments, case reports and case series have suggested that the approaches (e.g., CBT, SSRI) used to treat SAD without comorbid psychosis may also benefit patients with SAD.^{6,12,16} It seems important to keep in mind, however, that SSRIs may by themselves cause akathisia.¹⁷ Our patient's case and available evidence suggest that identifying and treating SAD in patients with schizophrenia may help promote recovery.

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